



UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
-----------------	-------------	----------------------	---------------------	------------------

10/005,131

12/05/2001

Geoffrey Goldspink

10103-004

8321

20583

7590

02/07/2008

JONES DAY

222 EAST 41ST ST

NEW YORK, NY 10017

EXAMINER

HAMA, JOANNE

ART UNIT

PAPER NUMBER

1632

MAIL DATE

DELIVERY MODE

02/07/2008

PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

BJ

Office Action Summary

Application No.

10/005,131

Applicant(s)

GOLDSPINK, GEOFFREY

Examiner

Joanne Hama, Ph.D.

Art Unit

1632

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 06 November 2007.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 31-35, 40-42, 51 and 97-99 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 31-35, 40-42, 51 and 97-99 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
 2. ☐ Certified copies of the priority documents have been received in Application No. _____.
 3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- | | |
|--|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892) | 4) <input type="checkbox"/> Interview Summary (PTO-413) |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948) | Paper No(s)/Mail Date. _____ |
| 3) <input checked="" type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08) | 5) <input type="checkbox"/> Notice of Informal Patent Application |
| Paper No(s)/Mail Date _____ | 6) <input type="checkbox"/> Other: _____ |

DETAILED ACTION

Applicant filed a response to the Non-Final Action of May 3, 2007 on November 6, 2007. Claims 1-30, 36-39, 43-50, 52-96 are cancelled. Claims 31, 32, 51, 97, 99 are amended.

Claims 31-35, 40-42, 51, 97-99 are under consideration.

Information Disclosure Statement

Applicant filed an Information Disclosure Statement (IDS) on November 6, 2007. The IDS has been considered. Applicant has also included in the filing a copy of an IDS filed July 31, 2002. It is noted that the July 31, 2002 has already been considered by the Examiner on October 27, 2004.

Withdrawn Rejections/Objections

Claim Objection

Applicant's arguments, see page 4 of Applicant's response, filed November 6, 2007, with respect to the objection of claims 31-35, 40-42, 51, 58-62, 67-69, 78, 97-99 have been fully considered and are persuasive. With regard to claim 31 (a) (ii) being objected for comprising non-elected subject matter (see restriction February 18, 2004), "myosin heavy chain promoter", Applicant has deleted the term. With regard to claim 31 (c) being drawn to the use of a viral strain, Applicant has deleted the term. The objection of claims 31-35, 40-42, 51, 97-99 has been withdrawn. With regard to the

objection of claims 58-62, 67-69, 78, the objection is withdrawn as the claims are cancelled.

35 U.S.C. § 112, 1st parag., New Matter

Applicant's arguments, see pages 4-5 of Applicant's response, filed November 6, 2007, with respect to the rejection of claims 58-62, 67-69, 78, 97, 98 have been fully considered and are persuasive. Applicant has cancelled claims 58-62, 67-69, 78 and thus the rejection as it applies to these claims is withdrawn. The rejection of claims 97, 98, as they depended on claim 58 is withdrawn as claim 58 is cancelled.

New/Maintained Rejections

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 31-35, 40-42, 51, 97-99 are newly rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This is a new matter rejection.

Upon further consideration, amendment to claim 31, filed December 29, 2005, raises issues of new matter. Claim 31 has been amended to include the phrase, "a metabolic disorder or condition related to an alpha-galactosidase A deficiency." This raises issues of new matter because the scope of what is to be treated (any disorder or condition related to alpha-galactosidase A deficiency) and what polynucleotide (any polynucleotide) was envisioned to be used to treat the disorder or condition was not taught in the specification. For example, Medin et al., 1996, PNAS, USA, 93: 7912-7922, teach that Fabry patients exhibit renal, cardio-, or cerebrovascular disease (Medin et al., abstract). However, nothing in the specification teaches what gene(s) were envisioned that could treat the symptoms seen in these organs. The specification generally indicates therapeutic products (specification, page 5, 2nd parag. under "Polynucleotide sequences of interest"), but the specification does not teach what therapeutic products are to be used to treat renal, cardio-, or cerebrovascular diseases associated with alpha-galactosidase A deficiency. Because it was not envisioned that particular nucleic acids were to be used to treat various conditions in various tissues affected in Fabry disease, the amendment to claim 31 is new matter.

Claims 31-35, 40-42, 97-99 remain rejected in modified form under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method for treatment of a metabolic disorder or condition resulting from an alpha-galactosidase A deficiency, said method comprising administering to a subject in

need thereof an effective, non-toxic amount of a pharmaceutical composition and a pharmaceutically acceptable carrier, said pharmaceutical composition comprises:

- a) an expression cassette operably linked to :
 - i) a myosin light chain enhancer,
 - ii) a viral promoter; and
 - iii) a polynucleotide sequence encoding alpha-galactosidase A, or
- b) a vector comprising said expression cassette,

does not reasonably provide enablement for

a method a method for treatment of a metabolic disorder or condition resulting from an alpha-galactosidase A deficiency, said method comprising administering to a subject in need thereof an effective, non-toxic amount of a pharmaceutical composition and a pharmaceutically acceptable carrier, said pharmaceutical composition comprises:

- a) an expression cassette operably linked to :
 - i) a myosin light chain enhancer,
 - ii) a viral promoter; and
 - iii) any polynucleotide sequence encoding a polypeptide of therapeutic use, or
- b) a vector comprising said expression cassette.

The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims, for reasons of record, May 3, 2007.

Applicant's arguments filed November 6, 2007 have been fully considered and they are persuasive in part.

With regard to the Office Action, May 3, 2007, pages 5-6, indicating that the claims are drawn to the treatment of the symptoms associated with alpha-galactosidase A deficiency, using any polynucleotide that encodes any therapeutic polypeptide (other than alpha-galactosidase A), the rejection as they apply to the claims remain. Applicant indicates that the specification provides guidance for use of the polynucleotide sequences of interest (Applicant's response, page 9, under "8. The Breadth of the Claims"). Applicant indicates that the specification provides guidance of using alpha-galactosidase (Applicant's response, pages 7-8, under "2. The Amount of Direction or Guidance Presented", under "3. The Presence of Working Examples", under "5. The State of the Art"). However, while Applicant indicates that the specification provides guidance for alpha-galactosidase A, this does not provide guidance for the breadth of symptoms (angiokeratomas, hypohidrosis, proteinuria, renal failure) that result from an alpha-galactosidase A deficiency and are treated by other therapeutic polypeptides (see also New Matter rejection, above). While Applicant's response is persuasive for using alpha-galactosidase to treat a disorder or condition related to an alpha-galactosidase A deficiency, the response is not persuasive for the full breadth encompassed by the claims, wherein the method treats particular symptoms of the disorder or condition, using other polypeptides. As such, the rejection as it applies to claim 51 is withdrawn; however, with regard to the claims for encompassing other nucleic acid sequences that encode peptides other than alpha-galactosidase A, the rejection remains.

With regard to the Office Action, pages 6-7, indicating that the claims were not enabled for gene therapy via injection of expression construct into muscle, the Examiner reconsiders the rejection and withdraws the rejection.

With regard to the Office Action, pages 7-8, indicating that the claims were not enabled for use of homologous sequences (claims 41, 42), the Examiner reconsiders the rejection and withdraws the rejection.

With regard to the Office Action, page 8, indicating that enzyme replacement therapy for Fabry disease was known to those skilled in the art, Applicant indicates that work by Sugimoto et al., 1995, Hum. Gen. Ther. 6: 905-915 teach that their retroviral vector system of expressing alpha-galactosidase A has usefulness in gene therapy of Fabry disease. Further Ohshima et al. 1997, PNAS, USA, 94: 2540-2544 demonstrate the correction of embryonic alpha-galactosidase deficient cells by transducing these cells with bicistronic multidrug resistance retroviruses containing alpha-galactosidase A cDNA (Applicant's response, page 8, under "5. The State of the Art"). In response, this is persuasive and the rejection as it applies to this issue is withdrawn.

For these reasons, the claims remain rejected.

Claims 31-35, 40-42, 97-99 remain rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention, May 3, 2007.

Applicant's arguments filed November 6, 2007 have been fully considered but they are not persuasive.

Applicant indicates that the instant claims are drawn to a method of treating the underlying cause of an alpha-galactosidase A deficiency and not the symptoms of the disease. The underlying case being a defect in alpha-galactosidase A gene resulting in deficiency of production of the corresponding enzyme. Applicant indicates that the specification provides extensive disclosure of all the elements in the claimed methods and that the specification provides working examples (Applicant's response, page 5). Applicant indicates that all the essential elements of the claimed invention are adequately described in the instant specification. Applicant reiterates that the instant claims are drawn to a method of treating the underlying cause of an alpha-galactosidase A deficiency and not the symptoms of the disease (Applicant's response, page 6). In response, this is not persuasive. While the specification provides guidance using wild type alpha-galactosidase A to treat Fabry disease, the specification does not provide guidance for treatment of specific symptoms associated with an alpha-galactosidase A deficiency, using peptides other than alpha-galactosidase A. While Applicant indicates that the claims are drawn to a method of treating the underlying case of an alpha-galactosidase deficiency, the claims, as written do not reflect what Applicant asserts. The claims are to treating any metabolic disorder or condition, using any therapeutic polypeptide. The rejection is maintained as the specification does not provide guidance of the various polypeptides envisioned to treat the metabolic disorder

or condition (e.g. angiokeratomas, hypohidrosis, proteinuria, renal failure) related to an alpha-galactosidase A deficiency. As such, the claims remain rejected.

It is noted that the rejection of claim 51 is withdrawn as the claim has been amended to "alpha-galactosidase".

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

Claims 31-35, 40, 51, 97, 98 are newly rejected under 35 U.S.C. 103(a) as being unpatentable over Medin et al., 1996, PNAS, USA, 93: 7917-7922, in view of Wolff et al., 1990, Science, 247: 1465-1468 (see IDS, filed November 6, 2007), in view of Davis et al., 1993, Human Gene Therapy, 4: 733-740 (see IDS filed November 6, 2007), in view of Goldspink et al., WO 94/28151, published December 8, 1994 (cited by Examiner, June 30, 2005), in view of Donoghue et al., 1988, Genes and Development, 2: 1779-1790 (see IDS filed July 31, 2002).

Medin et al. teach a retroviral construct comprising a nucleic acid sequence encoding full length alpha-galactosidase A and that the vector construct was used to infect AM12 cells. AM12 cells were used as virus-producing cells and as a source of recombinant alpha-galactosidase A enzyme (Medin et al., page 7917, 2nd col. under "Vector Construction"; see also page 7918, under "The Retroviral PG1alpha-Gal A

construct"). Medin et al. teach that alpha-gal secreted from AM12 infected cells could be used to treat cultured skin fibroblasts from Fabry patients (Medin et al., page 7918, under "Enzymatic Correction of Cells Obtained from Patients with Fabry Disease"). Medin et al. teach that treatment of Fabry disease was initially carried out by infusion of normal human plasma or partially purified preparations of alpha-galactosidase A to patients. The enzyme has a short half-life in circulation and the corrective activity was rapidly cleared (Medin et al., page 7921, 2nd parag. under "Discussion"). Medin et al. teach that their retroviral vector construct has applications to treat Fabry patients' cells (Medin et al., abstract).

While Medin et al. teach that a viral expression vector can be used to express alpha-galactosidase A in vitro, Medin et al. do not teach a plasmid vector comprising a nucleic acid sequence encoding alpha-galactosidase A, nor do they teach in vivo treatment.

Wolff et al. teach that injection of DNA directly into mouse skeletal muscle results in significant expression of the gene of interest within the muscle cells. Wolff et al. teach that expression of the protein expressed from the transgene was present in the muscle for at least 2 months. (Wolff et al., abstract and page 1465, 2nd col., 1st parag.). In their study, Wolff et al. teach that RSVL plasmid containing the firefly luciferase reporter gene exhibited a dose-response effect (i.e. ten times more vector resulted in ten times more protein being expressed), that the yield of recombinant protein was similar to that of fibroblasts, and that expression of luciferase was seen for at least 60 days (Wolff et al., page 1466). Wolff et al. teach that direct transfer of genes into

human muscle has clinical applications and can be used to ameliorate genetic disease by expression of the normal gene within muscle cells. Muscle would also be a suitable tissue for the expression of a transgene that would modify disease states in which muscle is not primarily involved (page 1467, 3rd col., 3rd parag. to page 1468).

Davis et al. compare the gene transfer ability of retroviral, adenoviral, and plasmid vectors. Davis et al. teach that plasmid DNA can be incorporated as well as or better than the genomes of either adenoviral or retroviral vector, regardless of whether the muscle was in mature state or undergoing regeneration (Davis et al., page 738, under "Potential for therapeutic application"). Davis et al. also teach that there are factors other than efficiency of transfer that make pure recombinant DNA the vector of choice for gene transfer into skeletal muscle. Injection of plasmid DNA did not have any deleterious consequences (e.g. immune rejection or sensitization). Further, production of plasmid DNA is easier and less expensive than for viral vectors, the purity can be controlled better than cellular or viral material, and plasmid vector can carry large genes as well as the required transcriptional control element (e.g. promoters and enhancers) (Davis et al., page 739, 1st col., 2nd and 3rd parags.).

It would have been obvious to one of ordinary skill in the art to use gene therapy to treat Fabry disease. An artisan would have arrived at the claimed method of injecting into muscle, a plasmid construct comprising the nucleic acid sequence encoding alpha-galactosidase A. Medin et al. teach that while introduction of plasma containing alpha-galactosidase A protein to treat Fabry patients was known, the effect of treatment, when using plasma, was short. Medin et al. teach that gene therapy would provide a Fabry

patient with a more sustained source of wild-type alpha-galactosidase A. Medin et al. teach an in vitro study demonstrating that a retroviral vector that expresses alpha-galactosidase A can be used to transform cells which express and secrete alpha-galactosidase A that treats human Fabry patient cells. While Medin et al. teach their retroviral system in vitro, they do not teach in vivo treatment and a plasmid vector. The combined teachings of Wolff et al. and Davis et al. provide guidance that gene therapy techniques using plasmid injected into muscle were well known in the art. Wolff et al. and Davis et al. teach that plasmids express well in muscle and are better than viral vectors because they do not induce immune response and plasmid constructs are easier and inexpensive to make. Given the teachings of Medin et al., Wolff et al., and Davis et al., an artisan would have taken the plasmid expression vectors of Wolff et al. or Davis et al. and substituted the reporter genes (lacZ in Wolff et al. and Davis et al.) with an nucleic acid sequence encoding alpha-galactosidase A (taught by Medin et al., who used the nucleic acid sequence in their retrovirus expression construct). An artisan would have then taken the plasmid construct and introduced it to muscle of a patient, wherein alpha-galactosidase A protein would be expressed.

With regard to claim 31 being drawn to the use of a myosin light chain enhancer, Goldspink et al. teach that a myosin light chain enhancer sequence can be used in an expression construct in muscle cell expression systems (C2 myoblasts and pCH101) and that constructs that comprise the enhancer element upregulated expression more than constructs that did not comprise it (Goldspink et al., page 12, Example 3). As such, it would have been obvious for an artisan to include the myosin light chain

enhancer, in order to arrive at an expression construct that expressed more transgene in muscle.

With regard to the plasmid being administered as a naked nucleic acid construct (claim 33), it is noted that Wolff et al. teach that DNA (plasmid) was injected directly into mouse skeletal muscles (Wolff et al., page 1465, 2nd col., 1st parag.).

With regard to the myosin light chain enhancer being a myosin light chain 1/3 enhancer (claim 35), it is noted that Goldspink et al. indicates that the enhancer was taught by Donoghue et al. and that Donoghue et al. teach that this enhancer is 1/3.

With regard to the promoter being a cytomegalovirus or herpes simplex virus promoter (claim 40), it is noted that Davis et al. teach expression of a plasmid construct in muscle, using a CMV promoter (Davis et al., page 734, under "Expression vectors"). While Wolff et al. teach introducing the plasmid expression construct, using a RSV promoter, it would be as obvious to use a CMV as a RSV promoter because both promoters are functionally equivalent.

Thus, the claims are obvious.

Claims 31, 41, 42, 99 are newly rejected under 35 U.S.C. 103(a) as being unpatentable over Medin et al., 1996, PNAS, USA, 93: 7917-7922, in view of Wolff et al., 1990, Science, 247: 1465-1468 (see IDS, filed November 6, 2007), in view of Davis et al., 1993, Human Gene Therapy, 4: 733-740 (see IDS filed November 6, 2007), in view of Goldspink et al., WO 94/28151, published December 8, 1994 (cited by

Examiner, June 30, 2005), in view of Treco and Selden, 1995, Molecular Medicine Today, 1: 314-321.

As discussed above, the combined teachings of Medin et al., in view of Wolff et al., Davis et al., and Goldspink et al. provide guidance for an artisan to arrive at a method for treating a metabolic disorder or condition related to an alpha-galactosidase A deficiency, wherein a subject is administered a vector comprising a polynucleotide sequence encoding a polypeptide is operably linked to a myosin light chain enhancer. While the combined teachings render claims 31-35, 40, 51, 97, 98 obvious, the teachings do not make obvious claims 41 and 42, wherein the expression cassette is flanked by genomic sequence.

Treco and Selden teach that specific alteration of cellular sequences, either by site-specific integration of exogenous DNA or by the correction of defective genes, can be accomplished by homologous recombination between transfected and cellular DNA. Homologous recombination is an event whereby sequences present on a transfected DNA molecule (the targeting DNA) first pair with endogenous cellular sequences with which they share a high degree of sequence similarity (the target DNA). Cellular enzymes catalyze the exchange of information between the transfected DNA and cellular sequences, resulting in the incorporation of DAN sequence information into the target site (Treco and Selden, page 317, 1st parag. under "Gene targeting"). In addition to homologous recombination being used to substitute one sequence for another (e.g. substituting the mutant nucleic acid sequence in the genome with a exogenously supplied wild type sequence), Treco and Selden teach that homologous recombination

can be used to insert a functional therapeutic gene into a specific chromosomal location (Treco and Selden, page 317, 2nd col., 2nd parag.).

Thus, given the combined teachings of Medin et al., Wolff et al., Davis et al., and Goldspink et al., an artisan would have arrived at a plasmid vector comprising an expression cassette, wherein the expression cassette comprises a polynucleotide encoding a polypeptide of therapeutic use operably linked to a myosin light chain enhancer and a viral promoter. An artisan would have then taken the teachings of Treco and Selden, flanked the expression cassette with nucleic acid sequences that are homologous to a particular sequence in the target genome, and inserted the expression cassette into the target genome, using homologous recombination.

With regard to claim 42 being drawn to using viral genomic sequence that flanks the expression cassette, it would have been obvious to use viral genomic sequence if an artisan wants to stably integrate the construct of claim 31 into the viral genome.

Thus, the claims are obvious.

Conclusion

No claims allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Joanne Hama, Ph.D. whose telephone number is 571-272-2911. The examiner can normally be reached Monday through Thursday and alternate Fridays from 9:00-5:00.

Application/Control Number:
10/005,131
Art Unit: 1632

Page 16

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Peter Paras, can be reached on 571-272-4517. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

Patent applicants with problems or questions regarding electronic images that can be viewed in the Patent Application Information Retrieval system (PAIR) can now contact the USPTO's Patent Electronic Business Center (Patent EBC) for assistance. Representatives are available to answer your questions daily from 6 am to midnight (EST). The toll free number is (866) 217-9197. When calling please have your application serial or patent number, the type of document you are having an image problem with, the number of pages and the specific nature of the problem. The Patent Electronic Business Center will notify applicants of the resolution of the problem within 5-7 business days. Applicants can also check PAIR to confirm that the problem has been corrected. The USPTO's Patent Electronic Business Center is a complete service center supporting all patent business on the Internet. The USPTO's PAIR system provides Internet-based access to patent application status and history information. It also enables applicants to view the scanned images of their own application file folder(s) as well as general patent information available to the public. For all other customer support, please call the USPTO Call Center (UCC) at 800-786-9199.

Joanne Hama
Art Unit 1632

